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CLINICAL EXAMINATION TECHNIQUES IN OTOTOLOGY

[OTOLARYNGOLOGY CLINICAL EXAMINATION SERIES]

This book discusses the nitty gritty of examining a patient with otological problem. This whole book has been written with focus on clinical examination techniques.

Foreword

This is the first book of Otolaryngology clinical examination series. This book is devoted to honing the clinical examination skills of a student in the field of otology. The other two books will be released shortly.

Students will find useful material in this book which will help them during their clinical and practical examination in otology.

Clinical photographs and illustrations are provided where ever necessary.

Mail in your comments to: <mailto:drtbalu@gmail.com>

Clinical examination techniques in otology

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Introduction:

Before proceeding with clinical examination a good history ¹ taking is a must. Without proper history taking it is not possible to come to a reasonably correct diagnosis by clinical examination alone. A good history taken is half the good work done. It is so important that venturing a diagnosis without it is like venturing into a dark forest without a torch. History as provided by the patient will give useful pointers / clues towards probable diagnosis. No clinical examination should be considered to be complete without eliciting proper history from the patient.

While eliciting history, the examiner should be really patient and allow the patient to talk without interrupting unnecessarily. Proper notes should be made during the patient's narration. If the patient is narrating multiple problems the examiner should listen, examine, interpret explain and then treat ².

Examiner should communicate with the patient in very simple language. The following facts should be borne in mind while eliciting history from a patient:

A patient may provide inaccurate history due to any of the following reasons:

- a. The question asked was not understood properly by the patient
- b. The patient did not know the answer to the question
- c. Patients commonly use un conventional words to describe their ailment
- d. The patient may want to please the examiner / kith and kin

History taking Golden rules:

The following are the rules that should be followed when eliciting history from a patient.

1. The patient should always be encouraged to speak
2. Patient's narration should not be cut off prematurely
3. Leading questions should not be posed to the patient very early during history taking
4. Previous medical history should be elicited with great care. Time should be spent doing this.
5. Importance should be given to the chronology of events as described by the patient

6. Always check inaccuracies in the patient's narrative without accusation.
7. Questions should be rephrased to check for contradictions
8. Always take into consideration failing memory of the patient

History taking should be done in the following order:

- a. Chief complaint: The patient should be encouraged to describe the chief complaint in his/her own words. If the patient sounds vague a pointing question like "what has made you to seek medical attention?" If the patient is listing out a series of problems then chronological sequence of these problems become important.
- b. Further description of the patient regarding the chief complaints – This should characteristically include the site pertaining to the complaint, character of the problem, and its intensity. Particular emphasis should be given to the morbidity caused by the problem.
- c. Chronology – The time of onset of the problem is vital to the diagnosis of the disorder. To stress on its importance the patient can even be asked a pointed question "When were you last normal / symptom free?". The exact date of onset of the complaint should be elicited.
- d. Acuity of the problem: The patient should be asked to describe when exactly the symptoms turned worse.
- e. Associated symptoms: Any associated symptoms should be sought

History taking in otology should include ³:

- Previous ear surgery
- Previous head injury
- systemic diseases like diabetes / hypertension
- Use of ototoxic drugs
- Exposure to noise during work
- Family h/o deafness
- H/O atopy / allergy

Symptomatology ⁴:

Common symptoms a patient with otological problems present with include:

1. Deafness
2. Discharge
3. Tinnitus
4. Pain
5. Vertigo

Deafness:

In clinical terms the symptom deafness is defined as any difficulty in hearing. It is also known as hard of hearing. This is actually one of the common otological symptoms a patient can present with.

While eliciting history of deafness from a patient the following factors should be taken into account:

Age of the patient: This is important because as one grows older there is natural degeneration of hair cells which are vital for acuity of hearing. High frequency hearing loss is seen in patients above the age of 60. This is known as presbycusis⁵. Classically these patients with presbycusis convey their inability to comprehend spoken words. They are able to hear the sound, but are not able to clearly comprehend its meaning.

Whether deafness is unilateral / bilateral:

Bilateral deafness is possible in patients with Noise induced hearing loss, Otosclerosis. In noise induced hearing loss⁶ deafness is sensorineural in nature. Pure tone audiometry will show high frequency sensori neural hearing loss.

Noise induced hearing loss: could be present in two forms⁷.

Temporary threshold shift: In this type hearing loss is transient. If the patient is removed from noisy environment hearing will improve. This is due to auditory fatigue due to exposure of loud noise. Temporary threshold shift exists in two forms:

Short term fatigue⁸:

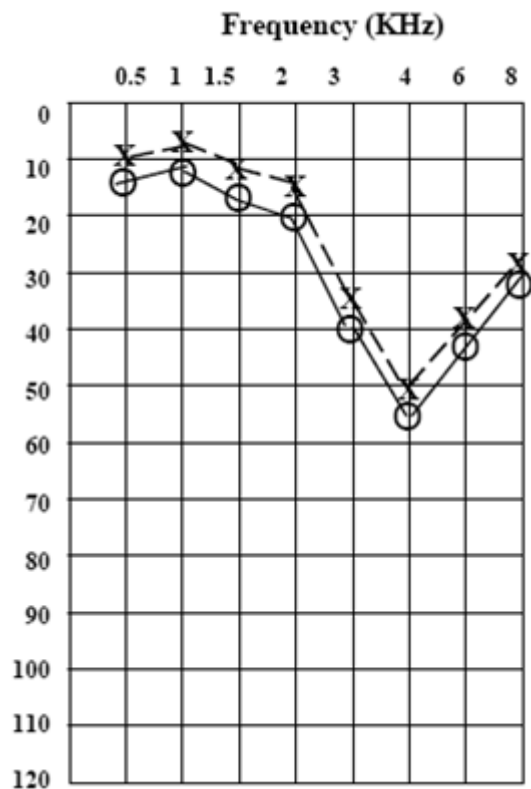
In this type of fatigue hearing recovery occurs within minutes. It is independent of the duration of noise exposure. Hearing loss is maximal for the frequency of noise exposed.

Long term fatigue⁹:

Recovery requires several minutes to days. This type of deafness is dependent on the duration of noise exposure.

Permanent threshold shift: In this type the hearing loss is permanent. This tends to occur in patients with temporary threshold shift who continue to be exposed to the offending noisy environment.

Noise induced hearing loss is the common cause of preventable deafness. This is included under workmen compensation act¹⁰.



Puretone audiogram showing bilateral noise induced hearing loss. Classically dip is seen at

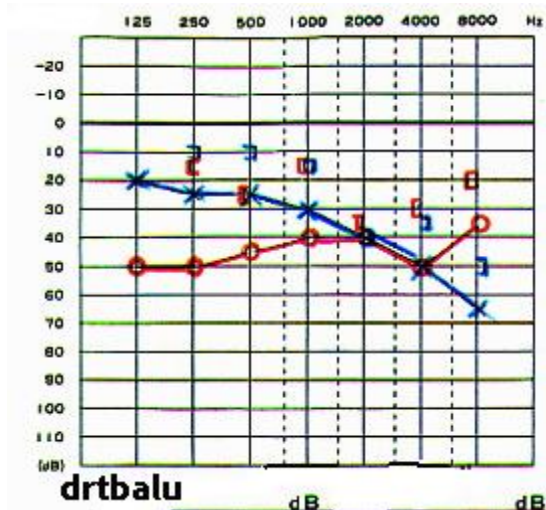
4 k Hz

Otosclerosis:

This is another cause of bilateral deafness. Commonly deafness is conductive in nature in this condition. In Otospongiosis¹¹ (active form of otosclerosis) deafness is sensori neural in nature. These patients reveal that they are able to hear better in noisy environment (Paracusis Wilisi)¹¹.

Pure tone audiometry in these patients shows bilateral conductive hearing loss. Typically audiogram in these patients reveal cookie bite pattern “Cookie bite audiogram”.

Another feature in an audiogram of a patient with otosclerosis is the presence of Carhart's notch. It is classically found in bone conduction audiograms of patients with otosclerosis. This is actually a dip centered around 2000 Hz.



Audiogram of a patient with otosclerosis showing Carhart's notch.

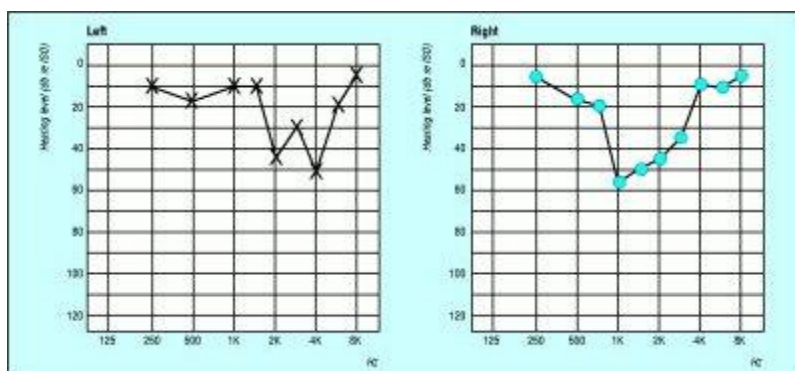


Figure showing cookie bite audiogram

Low frequency sensorineural hearing loss in patients indicates the probable presence of Meniere's disease. This should be viewed in the context of sensorineural hearing loss commonly causes high frequency hearing loss ¹².

Duration of deafness:

Deafness is acute when it is due to:

Trauma

Head injuries

When it is caused due to vascular causes

Deafness is chronic when it is due to:

Impacted wax

Acute / Chronic suppurative otitis media

Presbycusis

Progressive / sudden:

This is another important parameter which should be carefully gleaned from history of the patient. Sudden deafness is commonly seen in:

1. Sudden vascular insults
2. Head injuries
3. Traumatic injuries to ear drum and middle ear structures

Progressive deafness is seen in:

1. Accumulation of cerumen
2. Otitis media
3. Presbycusis
4. Noise induced hearing loss

Is it accompanied by tinnitus / vertigo?:

Deafness accompanied by tinnitus indicates early sensorineural hearing loss / Meniere's disease / Presbycusis. Vertigo commonly accompanies patients with Meniere's disease ¹².

Vertigo is defined as a sensation of unsteadiness / rotation. The commonest peripheral causes for vertigo are the diseases affecting the inner ear. It is always associated with tinnitus/ blocking sensation in the ear. Peripheral vertigo can be differentiated by central vertigo by its fatigability. In peripheral vertigo the vertigo tends to diminish with time because the higher centre learns to adjust with the problem. It is always positional. The patient will have to assume the provoking position for vertigo to manifest. Vertigo due to Meniere's disease is self-limiting and short lived. It never lasts for more than a day after which the patient gradually improves. Peripheral vertigo is always associated with horizontal nystagmus, which is again fatiguing, whereas central nystagmus due to cerebellar pathology manifests with rotatory / vertical nystagmus. They also show other positive cerebellar signs like past pointing, dysdiadokokinesis etc.

Vertigo alone is seen in patients with:

1. Vestibular neuronitis
2. BPPV
3. Labyrinthitis

Differences between central and peripheral vertigo ⁴:

Central vertigo	Peripheral vertigo
Not fatigable	Fatigable
Not positional	Always positional
Vertical / rotatory nystagmus	Horizontal nystagmus
Cerebellar signs present	Cerebellar signs absent

Tinnitus:

Tinnitus is defined as hearing abnormal sounds in the ear. It can be classified into objective tinnitus and subjective tinnitus. Objective tinnitus is the one which is heard by both the examiner and the patient e.g. palatal myoclonus. Subjective tinnitus is heard only by the patient. Even a simple problem like impacted wax can cause subjective tinnitus by the process of amplification of endogenous sound (internal milieu sounds of the body like the

sound of circulating blood, contraction of muscle etc.) Commonly tinnitus (subjective) in the absence of impacted cerumen indicates early sensori neural hearing loss. This is caused by damage to hair cells of the cochlea. The damage could be due to the adverse effects of medicines like those belonging to the group of antibiotics, diuretics or cytotoxic drugs. Tinnitus associated with hearing loss is commonly a manifestation of Meniere's syndrome. Tinnitus in this syndrome is roaring in nature and resolves within a day. It is also associated with giddiness.

Tinnitus in a patient with otosclerosis is an indication of active disease. These patients have active foci of otosclerosis. A separate term is used to identify these patients i.e. Otospongiosis. Surgery if performed during this phase carries an immense risk of sensorineural hearing loss.

Discharge: Ear discharge is one of the common problems that brings the patient to the doctor. Before examining the patient a detailed history regarding

1. Duration of the discharge
2. Quantity of discharge
3. Quality of discharge
4. Aggravating & relieving factors must be sought for.

If the duration of discharge is short then acute conditions must be borne in mind. Common acute conditions which can lead to ear discharge are

1. A.S.O.M. - here the discharge is Serosanguinous in nature (blood tinged), preceded by an episode of severe ear pain, pain subsides as soon as discharge starts, preceding episodes of upper respiratory infection.
2. Otomycosis - common fungi affecting the external canal are candida and aspergillus fumigatus. Candida gives a curdy appearance in the external ear canal. In a dried up state it could appear like a cotton wool. Aspergillus fumigatus appears as a black color patches in the external auditory canal. These patients have ear discharge mostly just wetness, not profuse in nature, associated with intense itching.
3. C.S.F. Otorrhoea - The discharge is watery in nature, there is absolutely no mucoid elements in the discharge. This clear discharge starts when the affected ear assumes a dependent position. Biochemical analysis of this discharge will show that it contains glucose which is normally absent in purulent ear discharges.

Bedside test - One useful bedside test for CSF otorrhoea is Handkerchief test. If the secretion is mopped with a handkerchief and allowed to dry, there will be stiffening of the handkerchief if the discharge is from the middle ear because of the presence of mucous, if the discharge is CSF there is no stiffening seen.

Most sensitive diagnostic test is estimation of *Beta 2 transferrin* in the secretions. Beta 2 transferrin is seen only in the CSF and is absent in other types of discharges.

Another important factor in the history taking is asking for the quantity of discharge. The normal middle ear can hold up to 0.5 ml of fluid. On successful mopping it would take at least a couple of hours for pus to reaccumulate. If the discharge is profuse the following conditions must be borne in mind: chronic mastoiditis, mastoid reservoir, extra dural abscess. Of these three in extra dural abscess the discharge is so profuse the external canal fills up with pus immediately after mopping. The presence of mastoiditis or mastoid reservoir can be ruled out by looking out for tenderness in the mastoid tip area. In children with well pneumatized mastoids the pus may cause erosion of the outer cortex and present as a collection just under the mastoid periosteum. This condition is known as *sub periosteal abscess*. If the ear discharge is scanty and foul smelling osteitic reaction due to infection must be suspected. This is frequently caused by the presence of cholesteatoma in the middle ear cavity associated with bone erosion.

The quality of discharge may range from:

Mucoid - common in CSOM

Mucopurulent - common in CSOM associated with mastoiditis

Serous - Common in ASOM

Serosanguinous - ASOM and otitis externa

Watery - CSF otorrhoea

Ear discharge associated with pain is a feature of:

1. Otitis externa
2. Acute otitis media

In acute otitis media pain subsides after discharge begins.

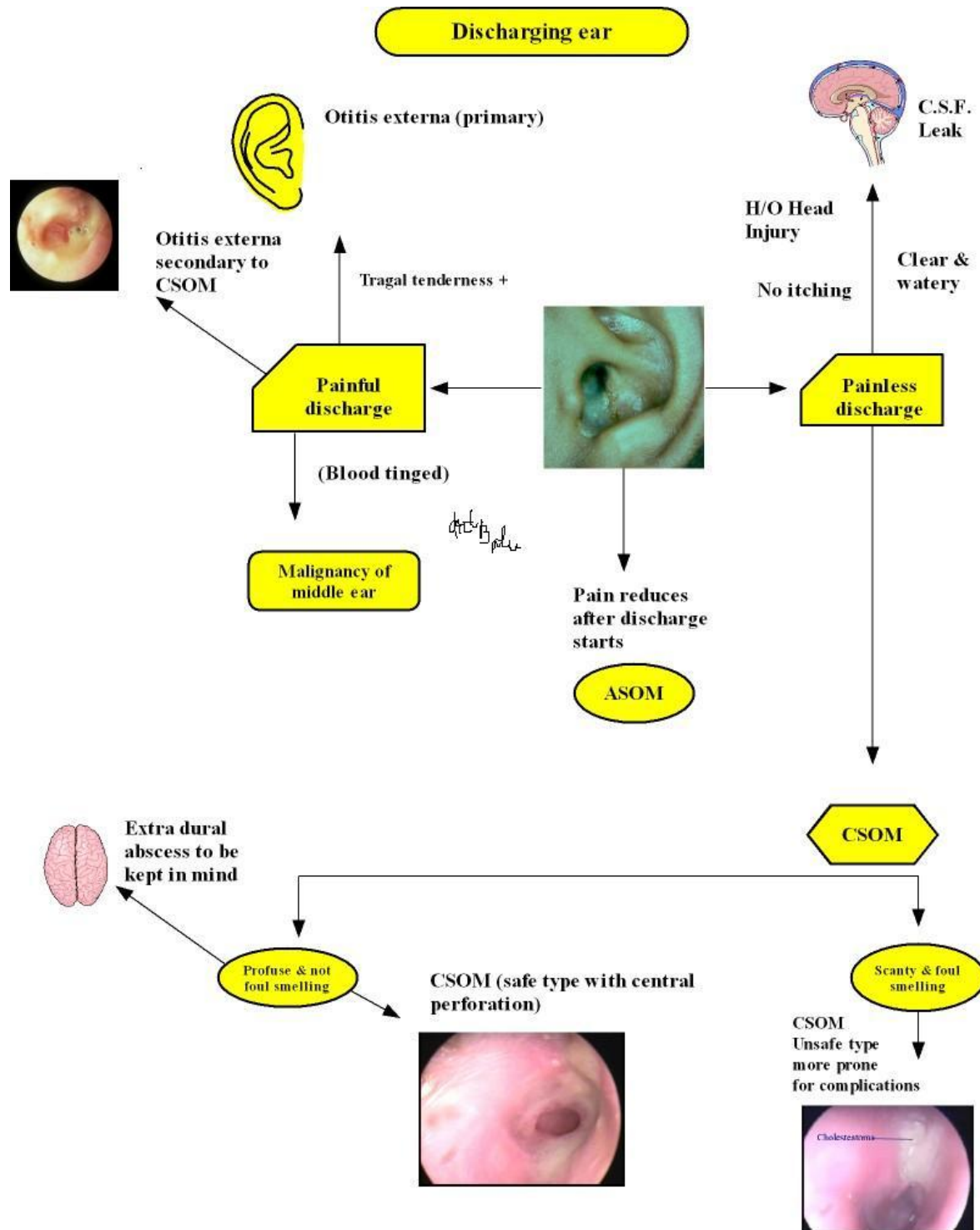


Figure showing differential diagnosis of a discharging ear

Pain in the ear is known as otalgia. Otolgia can be Primary or Referred.

Primary otalgia is pain in the ear due to ear pathology¹⁴. On the contrary referred Otolgia is not associated with ear pathology and hence is also known as secondary of non otogenic otalgia. Talmud 5th century AD physician¹⁴ prescribed juice of Kidney (urine) as treatment for otalgia.

Pain: is one of the common complaints in patients with ear problem. Pain in the ear can arise from 2 sources, pain due to problems confined to the ear, and referred otalgia i.e. pain that is referred to the ear from a problem arising from other areas, i.e. pain associated with tonsillar infection has a propensity to radiate to the ear due to common nerve supply i.e. glossopharyngeal nerve. Pain due to inflammation in the external ear is intense and is associated with swelling of the external auditory canal. This can be differentiated from pain arising from middle ear inflammation by the presence of tenderness on pressing the tragus. This sign is known as the *tragal sign*. Tragal sign is negative in otalgia due to middle ear causes. Pain due to mastoiditis (inflammation of mastoid air cells) can be differentiated from pain due to otitis externa by the presence of three point tenderness. Three point tenderness is elicited by using the middle finger to apply pressure over the well of the concha, index finger is applied over the mastoid process, and the thumb is used over the mastoid tip. The pressure over the well of the concha indicates tenderness over the antral area, tenderness over the mastoid process indicates the presence of mastoiditis, and tenderness over the tip of the mastoid process indicates inflammation and thrombosis of mastoid emissary vein.

Innervation of ear:

Knowledge of innervation of ear plays a vital role in understanding the causes of otalgia. Sensory innervation of the ear is via cranial and cervical nerves. There is considerable overlap between different nerves that provide sensation to the pinna. This is due to its complex embryological origin.

The auriculotemporal branch of mandibular nerve innervates the anterior portion of pinna, tragus and anterior wall of external auditory canal.

The well of the concha and the posterior wall of the external auditory canal are innervated by 7th 9th and 10th cranial nerves. Posteromedial aspect of auricle and the patch of skin over mastoid process is also innervated by this cranial nerve complex. Of course there may be significant individual variations in the actual sensory areas innervated by this cranial

nerve complex. Rest of the pinna is innervated by the greater auricular nerve. This greater auricular nerve is derived from the cervical plexus with contributions from C2 and C3.

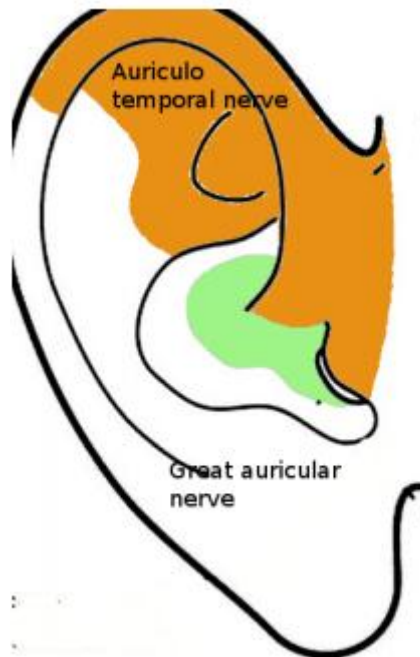


Figure showing innervation of lateral wall of pinna

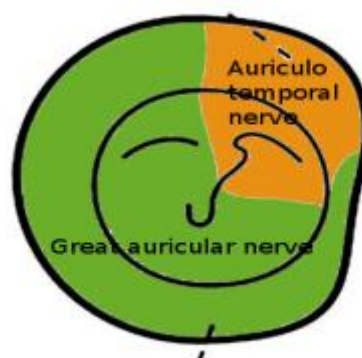
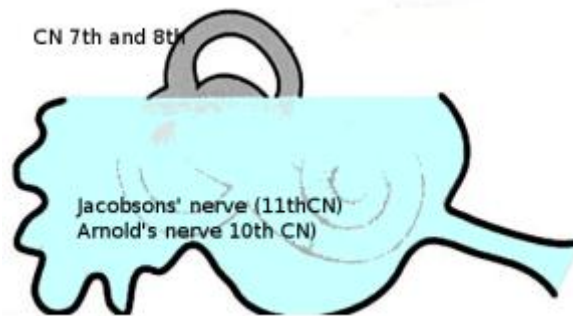


Figure showing innervation of external auditory canal



Innervation of Middle ear:

The middle ear receives sensory afferents primarily from the glossopharyngeal component of tympanic plexus. These sensory afferents are largely formed by the Jacobson's branch of glossopharyngeal nerve. The facial nerve receives sensation from the medial portion of pars flaccida and pars tensa by way of its chorda tympani branch.

Innervation of inner ear:

The inner ear is supplied by vestibulocochlear nerve. This nerve doesn't have pain fibers. Hence significant inner ear pathology can develop without pain. These nerves are sensitive to stretch hence in patients with endolymphatic hydrops there may be fullness of the membranous portion of inner ear which is perceived as pain by the patient.

Primary otalgia:

Is defined as pain due to pathology in the ear. This can be divided into causes involving the external ear, middle ear and inner ear.

External ear causes of otalgia:

Otitis externa is the most common cause of unilateral pain in the ear. This is due to infection of the external auditory canal. This condition is really a very painful one. Otitis external can be classified into:

1. Acute diffuse otitis externa (commonly caused by bacteria)

2. Acute localised otitis externa (commonly furuncle)
3. Chronic otitis externa
4. Eczematous otitis externa
5. Fungal otitis externa
6. Malignant otitis externa

Predisposing factors for otitis externa:

Under normal conditions the skin lining the external auditory canal is well protected by its self-cleansing mechanism. In diseased conditions several factors may come into play in the pathogenesis of otitis externa.

1. Absence of cerumen: The cerumen plays an important role in the protection of the external canal. It protects the external canal from moisture. It also has anti-bacterial properties which help in the protection of the external canal. The cerumen also lowers the pH of the external canal making it difficult for the bacterial pathogens to colonize.
2. Removal of cerumen by ear buds: is one of the common causes of otitis externa. The act of removal traumatises the skin lining of the external canal making it vulnerable to infections.
3. Frequent exposure to water: external canal when constantly bathed in water loses its ability to protect itself. The presence of water macerates the skin lining of the external canal and also increases the pH of the external canal making it more favourable for bacterial colonisation. This condition is common in swimmers.

Acute diffuse otitis externa:

This is also known as the swimmers ear. This is an inflammatory condition involving the external canal in a diffuse manner. This condition is common in swimmers because of the propensity for the external canal to be exposed to water for long durations. This exposure leads to maceration of the external canal skin, and also lowers the pH of the external canal providing an environment favourable to infections.

Main symptoms:

1. Itching in the external canal
2. Tenderness on palpation

3. Aural fullness rarely occur due to the reduction in size of the external canal lumen due to oedema

4. Rarely stenosis of the external canal can occur causing accumulation of debris and secretions.

Common signs:

1. Erythema of the external canal

2. Oedema of external canal

3. Secretions from the external canal (weeping canal)

4. Pain on mastication

5. Pulling of helix in a postero superior direction cause pain

6. In advanced cases fever and lymphadenopathy may occur (pre and post auricular nodes may be involved)

Stages of acute diffuse otitis externa: (Senturia)

Preinflammatory stage: is characterised by intense itching, oedema and sensation of fullness in the ear.

Inflammatory stage: may be divided into mild, moderate and severe.

Mild acute inflammatory stage: here the cardinal features are increased itching, pain, mild erythema and oedema of the external canal skin. At later stages exfoliation of skin with minimal amount of cloudy secretions may be seen in the external canal.

Moderate acute inflammatory stage: in this stage the itching and tenderness of the external canal intensifies. The external canal is narrowed due to oedema and accumulation of epithelial debris.

Severe acute inflammatory type: In this stage pain becomes intolerable to such an extent the patient may refuse to eat; the lumen of the external canal becomes totally obliterated due to oedema and accumulated epithelial debris. Otorrhoea may become purulent. In addition regional nodes may also be involved.

Infections from the external canal may involve the parotid gland via the fissure's of santorini. Common organisms involved: *Pseudomonas aeruginosa* and *staphylococcus aureus* are commonly cultured from the external canal of these patients. The normal commensals like *staphylococcus epidermidis* and *corynebacteria* are conspicuously absent.

Acute localized otitis externa:

This condition is otherwise known as furunculosis or circumscribed otitis externa. This is a localised infection usually found to involve the lateral 1/3 of the external canal. It also has a propensity to involve the posterior superior aspect of the external canal. This is caused due to obstruction of the apopilosebaceous units found extensively in this area.

Trauma to skin in this area followed by infection is commonly attributed cause.

The organism responsible is commonly staph aureus.

Symptoms:

1. Localised pain
2. Localised itching
3. Purulent discharge if the abscess ruptures
4. If oedema or abscess occludes the external canal hearing loss can occur.

Signs:

1. Erythema of the skin
2. Localised abscess formation

Chronic otitis externa:

This is a chronic infection / inflammation involving the skin lining of the external canal. There is thickening of the skin lining of the external canal due to persistent low grade infection / inflammation.

Symptoms:

1. Unrelenting pruritus
2. Mild pain
3. Presence of dry skin in the external canal

Signs:

1. Asteatosis (lack of cerumen)

2. Hypertrophic external canal skin
3. Presence of dry flaky skin in the external canal
4. Mild tenderness on ear manipulation
5. Rarely muco purulent otorrhoea

Cultures from the external canal of these patients are highly unreliable because they would have been using various antibiotic drops to surmount the problem.

Eczematous otitis externa:

This condition includes various dermatologic conditions involving the skin of the external canal. It may range from atopic dermatitis, contact dermatitis, seborrheic dermatitis, neuro dermatitis, infantile eczema etc.

This condition is characterised by intense itching, in fact this could be the only complaint of the patient. On examination, erythema of the external canal skin may be seen. There may also be associated scaling and oozing from the canal skin.

Fungal otitis externa:

This is the commonest type of otitis externa in tropical countries. This condition is associated with increased ear canal moisture, or following treatment of otitis external by prolonged use of topical antibiotics. The protective cerumen layer is absent in these patients. This condition is more common in diabetics.

Symptoms:

1. Intense itching
2. Pain when otitis externa is coexistent
3. Blocking sensation due to the presence of fungal balls

Signs:

1. Inflamed external canal skin
2. External canal tenderness
3. Fungal debris (black in case of aspergillus and white in the case of candida).

Invariably the infection is mixed type.

Malignant otitis externa:

This rare but sinister form of otitis externa is known to affect elderly diabetics.

This condition is caused by pseudomonas infection of the external ear. These patients have a unique nocturnal deep boring type of pain.

The patient gives history of trivial trauma to the external canal. Granulations can be seen at the junction of bony cartilaginous portion of the external canal.

This condition can cause complications like facial nerve involvement, and spread to the intracranial structures.

Chronic Myringitis:

This is defined as loss of tympanic membrane epithelium for more than a month in the absence of middle ear pathology. The etiology of this condition remains largely unknown. It may follow acute infections, trauma or previous ear surgery. It is often seen without any of these so called predisposing conditions.

The common presenting symptom is otalgia with associated otorrhoea, hearing loss, tinnitus, aural fullness and pruritus.

Otalgia in these patients is mediated by sensory afferents from the external auditory canal and lateral portion of ear drum. Granulation tissue may be seen in the external auditory canal and lateral surface of ear drum. Identification of this condition is vital as it would avoid unnecessary tympanomastoid surgery.

This is purely a clinical diagnosis and can be managed by avoiding aural manipulation. Intermittent use of topical antibiotics and steroids may help.

Bullous myringitis / Bullous otitis externa:

This condition is characterised by acute infection of ear drum / external auditory canal producing intraepithelial fluid collection. These patients commonly present with bullae in the lateral surface of ear drum (Bullous myringitis) or over the external auditory canal (Bullous otitis externa). These patients classically present with severe otalgia associated with serous / bloody discharge.

Middle ear causes of otalgia:

Acute otitis media:

Acute otitis media is one of the common middle ear causes of otalgia. This condition is common in children. It is caused by Eustachian tube block causing pent up secretions to accumulate in the middle ear cavity. Pain gets relieved when the ear drum perforates and starts to drain the middle ear cavity. Children are commonly affected because of their short,

wide and straight Eustachian tube. In patients with intractable pain then Myringotomy will have to be resorted to. Majority of them will respond well to oral antibiotics, analgesics and nasal decongestants.

Eustachian tube dysfunction:

This is one of the common middle ear causes of otalgia. Patients with eustachean tube dysfunction may have otalgia even in the absence of otitis media. This occurs because of the inability of middle ear to equalize with atmospheric pressure resulting in distortion to the middle ear mucosa and ear drum. Patients with retracted drum may experience acute ear pain when middle ear equalization manages to peel the retracted drum away from the promontory.

Otitis barotrauma:

This is caused due to descent in an aircraft which has not been pressurized / in air travellers with pre-existing Eustachian tube dysfunction. Since there is failure of equalisation due to inability of Eustachian tube to open normally the superficial blood vessels of middle ear mucosa ruptures leading on to hematoma formation. This is an acute painful condition. In some patients secretory otitis media may also be caused.

In these patients the middle ear fluid is believed to be transudate in nature, Whereas in secretory otitis media due to infection the middle ear fluid resembles exudate.

These patients should be treated with antibiotics (systemic), analgesics, and nasal decongestants. They are also encouraged to perform repeated valsalva maneuver in order to open up their Eustachian tubes.

Herpes zoster oticus (Ramsay Hunt syndrome):

This syndrome is characterised by otalgia, lower motor neuron type of facial palsy and the characteristic rash seen in the pinna / external auditory canal. Otalgia in these patients is caused by irritation / inflammation of the affected cranial nerve (7th nerve in this case). This condition is caused by Herpes infection.

Relapsing polychondritis:

This uncommon autoimmune disease involving the cartilage of pinna and external auditory canal causes intense otalgia and finally leads to fibrosis. Cartilage tissues which contain high quantities of glycosaminoglycans are commonly affected. Incidentally cartilage of pinna is rich in glycosaminoglycans. Other tissues containing high amounts of proteoglycans can also be involved (eg. Eyes, hear and inner ear). This condition is often misdiagnosed as being infectious in nature. Unlike auricular cellulitis the lobule of the pinna is spared in these patients as it is devoid of cartilage tissue. Diagnosis is made by biopsying

the tissue involved. These patients benefit from steroid and immunosuppressant administration.

Primary neoplasms arising from skull base / pinna / external canal:

Can also cause otalgia. Lesions originating from the skin covering of pinna include squamous cell carcinoma, basal cell carcinoma and melanoma. Ceruminous glands of external auditory canal can give rise to malignant lesions (adenocarcinoma). Other miscellaneous causes of primary otalgia include:

Trauma

Hematoma

Seroma

Frost bite

Burns / thermal injuries

Otalgia with indurate oedematous pinna should arise suspicion of cellulitis.

Chondrodermatitis nodularis chronica helicus:

This is an inflammatory nodule seen over helix of pinna with otalgia. This is commonly seen in males. It is a benign appearing tender lesion which can be treated with local excision and steroid infiltrations.

Secondary / Referred otalgia:

This is due to complex innervation of the ear. Noxious stimuli from remote areas of head and neck can manifest with otalgia due to the shared anatomic innervation of the ear. The causes for secondary otalgia are extensive and numerous. The three logical sites for initial attention in a case of referred otalgia are temporomandibular joint, entire neck and teeth. Among other probable causes head and neck malignancy should be excluded.

Temporomandibular joint syndrome:

In this condition the patient has difficulty / pain during mastication. These patients also manifest with aural fullness, tinnitus and vertigo. This syndrome goes by the name Costen syndrome. It was Costen who described this condition in precise detail in 1934. The symptoms associated with this syndrome are supposed to arise from posterior displacement of condyle of mandible, which leads to compression of auriculo temporal branch of trigeminal nerve of the chorda tympani branch of facial nerve.

Temporomandibular joint should be examined in all patients with otalgia. Focus should be on joint and muscles of mastication. It should also be seen whether careful palpation of joint structures reproduces otalgia.

Pterygoid muscles should be palpated intraorally in order to rule out spasm of the muscle.

Mandible should be distracted laterally to assess pain during various movements.

Presence of click / crepitus should be sought while performing this movement test.

Oral cavity of these patients should be examined with emphasis on identification of malocclusion which could cause excessive strain on the temporomandibular joint and its muscles.

Infections causing otalgia:

Infections that cause referred otalgia include:

1. Tonsillar infections (i.e. Quinsy, tonsillitis) via the glossopharyngeal nerve
2. Mumps parotitis – due to stretching of the sensitive parotid fascia via trigeminal nerve
3. Rarely sinus infections also can cause otalgia
4. Dental infections like tooth decay may cause referred otalgia

Eagle syndrome:

This is another potential cause of referred otalgia. This syndrome is defined as otalgia, facial pain, and throat pain secondary to elongated styloid process / ossified stylohyoid ligament.

Pain in Eagle's syndrome could be due to:

1. Direct compression / irritation of trigeminal, facial, glossopharyngeal and vagus nerves.
2. Direct compression / irritation of carotid vessels (Carotidynia)
3. Inflammation of tendinous part of stylohyoid ligament
4. Infection of styloid process and adjacent tissue (styloiditis)

Gastro oesophageal reflux disease:

A large number of disorders may be caused by gastro oesophageal reflux. It can cause otalgia by irritating the upper aero digestive tract in the sensory distribution of glossopharyngeal and vagus nerves. Sometimes reflux of acid contents from the stomach

may irritate the pharyngeal end of Eustachian tube causing acute otitis media. This GERD induced otitis media is common in children and toddlers.

Neuralgias:

The extensive innervation of the ear predisposes it to neuralgias. Neuralgic pain is usually brief, lancinating and episodic in nature. This type of pain is self-limiting and needs only reassurance. Neuralgias involving trigeminal, geniculate, glossopharyngeal and sphenopalatine ganglia manifest as otalgia.

Neoplasms:

Tumors involving various sites of head and neck can present with otalgia.

Tumors involving the anterior portion of the tongue may cause otalgia via the involvement of chorda tympani branch of facial nerve.

Nasal and paranasal sinus malignancies may cause otalgia secondary to Eustachian tube dysfunction or direct neural involvement. Direct neural involvement will cause otalgia through afferents from posterior lateral nasal nerves by way of sphenopalatine ganglion associated with the second division of trigeminal nerve.

Hypopharyngeal tumors especially those involving the pyriform fossa can cause otalgia via the vagus nerves.

Tonsillar malignancies may cause otalgia via the glossopharyngeal nerve.

The cervical plexus covers a large surface area of skin extending from the posterior aspect of the auricle on the mastoid to the lateral neck and thyroid. This explains referred ear pain in inflammations / malignancies of thyroid.

Lesions arising from infratemporal fossa can cause referred otalgia due to involvement of Arnold's nerve or Jacobson's nerve.

Cervical arthritis as a cause of referred otalgia:

Otalgia can be caused due to occipital nerve root irritation from cervical osteoarthritis. Spasm involving the cervical muscles can cause significant bilateral otalgia.

Otalgia can also be caused due to intracranial lesions due to stretching of Dura.

Inspection:

This is proceeded from outside to inside. (From pinna to ear drum). In the presence of perforation of ear drum a glimpse of middle ear mucosa could be seen.

Pinna:

The external ear is inspected with the following in mind:

1. size & shape of the pinna
2. Presence of tags / preauricular sinuses / pits
3. Evidence of trauma to pinna
4. Skin condition of pinna & external auditory canal
5. Evidence of previous surgery / presence of scars in the post aural / end aural region
6. Discharge from the external canal
7. Neoplastic lesions of pinna



Figure showing Microtia / skin tags



Figure showing preauricular sinus



Figure showing furuncle of external auditory canal



Figure showing otophyma

Examination of ear drum:

The ear drum can be examined using an otoscope. The pinna should be grasped between the index finger and thumb and is pulled postero superiorly. This maneuver straightens the external canal bringing the ear drum into full view. This maneuver should be done only in adults. In infants the pinna must be pulled posteriorly and downwards in an effort to straighten the external canal. This is because of the fact the bony portion of the external canal is not fully developed in infants.



Figure showing the procedure to straighten the external canal in an adult

The use of Grubber's aural speculum itself is sufficient to straighten the external canal. The status of the canal skin / presence or absence of discharge is noted. The whole of the ear drum is visualised by tilting and moving the otoscope in various directions.

The ear drum is roughly oval in shape and about 1 cm in diameter. Normal ear drum is pearly white in color. The following structures of ear drum are visualised:

1. Attic area
2. Pars tensa
3. Cone of light
4. Handle / lateral process of malleus

Rarely the following structures also can be seen:

Long process of incus

Head of stapes

Promontory

Eustachian tube orifice

Perforations any must be identified, its position clearly documented. Through the perforation the status of the middle ear mucosa must be observed and documented. Presence of tympanosclerotic plaque (chalky mass over the ear drum) is an indicator of previous ear disease.

The cone of light must be observed for any distortion. Cone of light is absent in perforated ear drums, is distorted in retracted ear drums. It is also distorted when the ear drum is bulging as in the case of Acute otitis media.

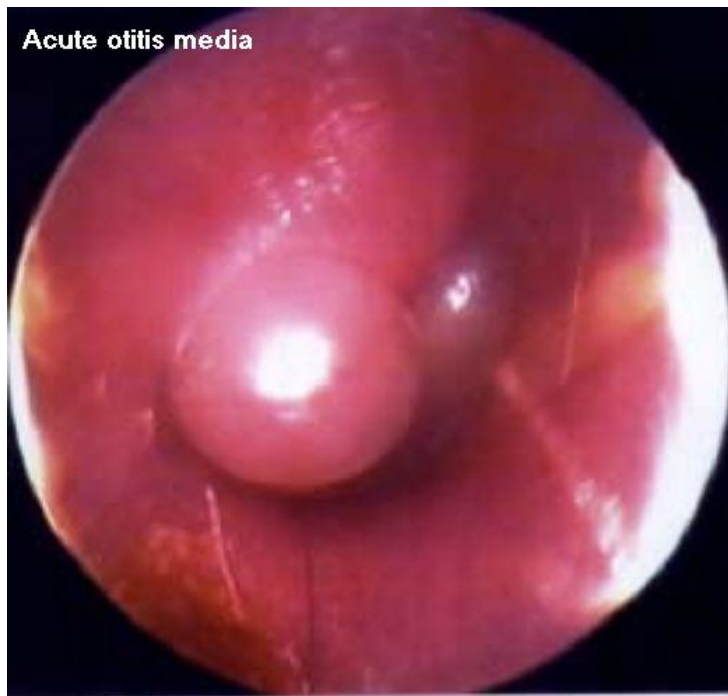
The color of the ear drum must also be noted:

Red drum - is seen in acute otitis media, glomus jugulare

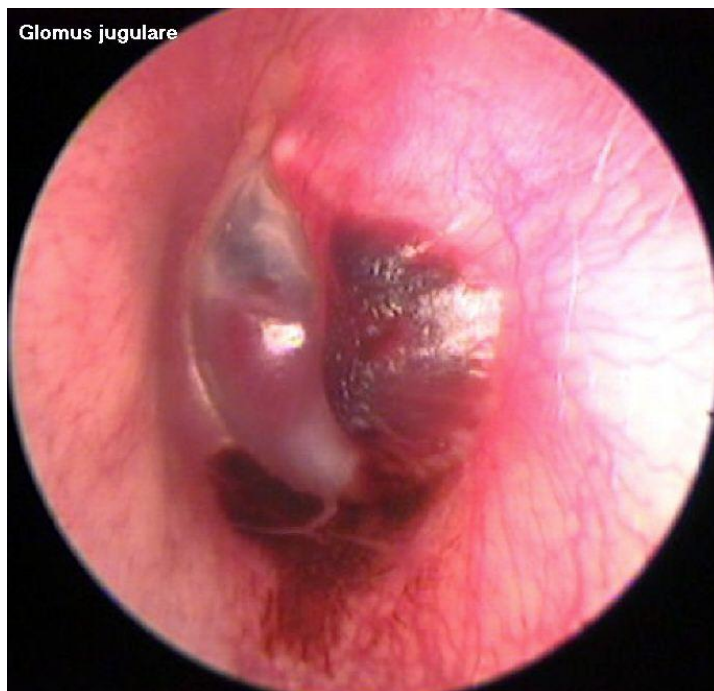
Blue drum - is seen in haemotympanum, secretory otitis media

Flamingo drum - is seen in otospongiosis

Mobility of the ear drum must be tested using a pneumatic otoscope, or a siegel's speculum. The mobility of the ear drum is restricted in adhesive otitis media.



Acute otitis media showing Red drum



Glomus jugulare causing red drum

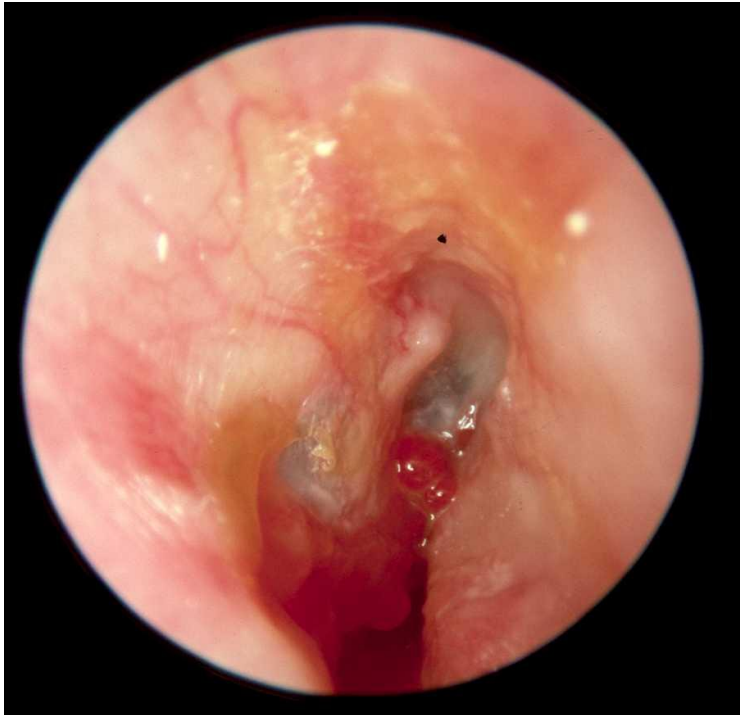


Figure showing myringitis granulosa

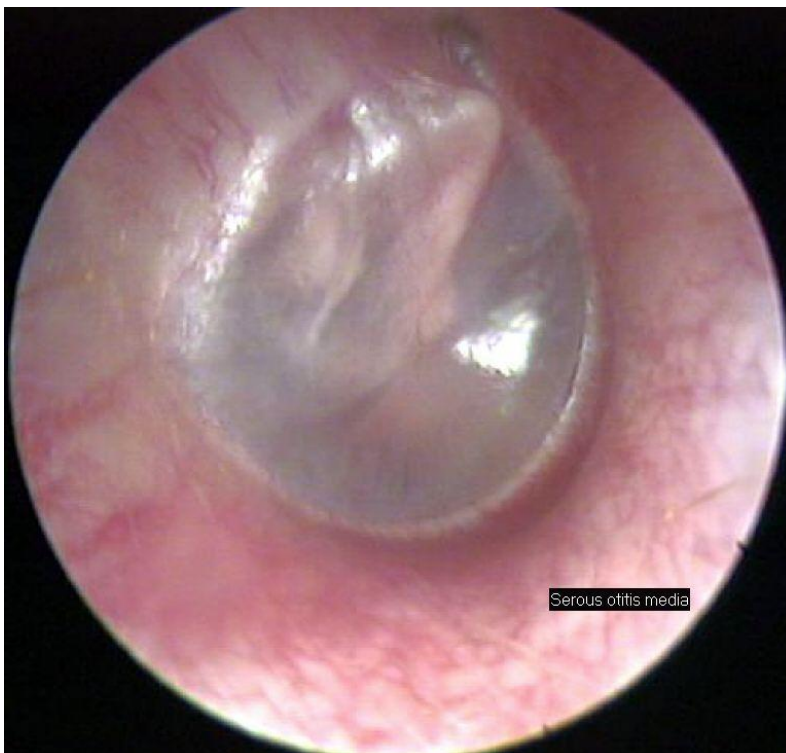
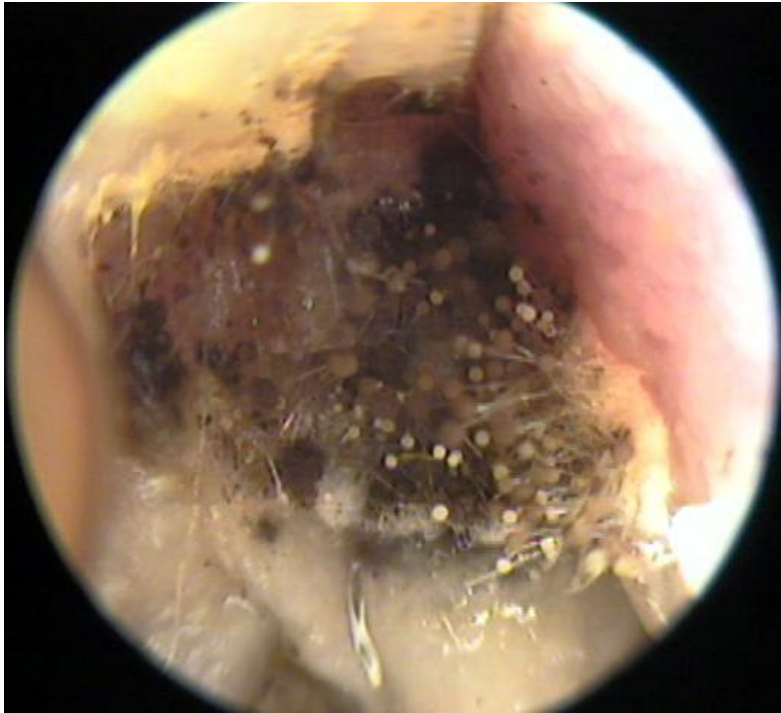


Figure showing secretory otitis media (Blue drum)

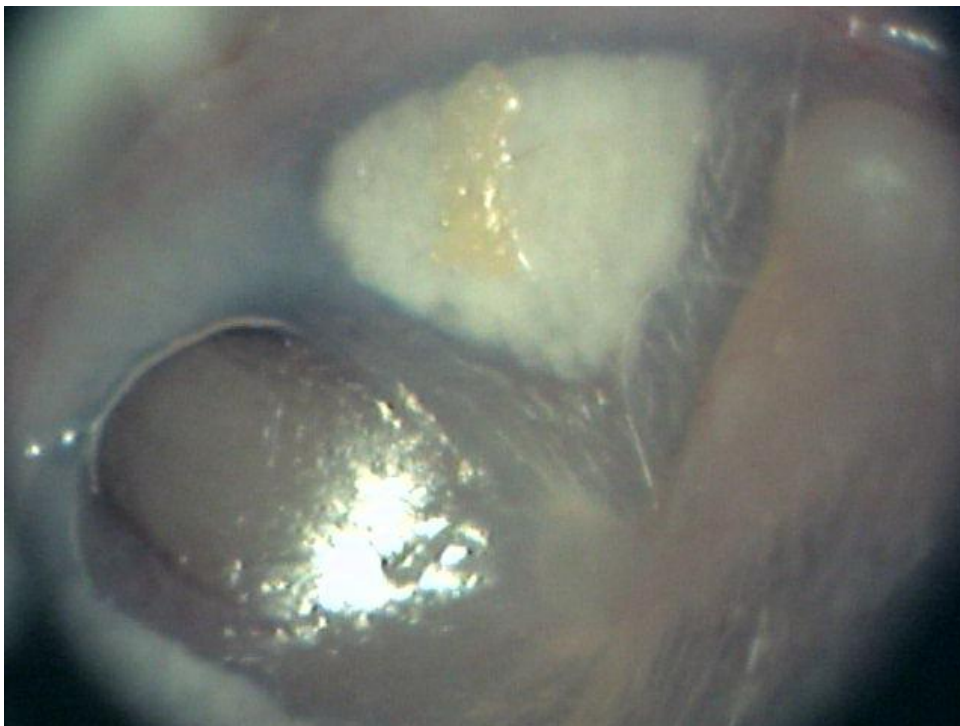


Otoscopic view showing cotton wool patches studded with black spots. This classic feature is seen in mixed fungal infections of external auditory canal. Whitish patches are caused by candida and black specs are caused by *Aspergillus niger*.

These patients classically present with blocked ear with intense itching. This itching sensation causes them to keep cleaning their ear with cotton buds which could traumatize the tender skin of external auditory canal. This trauma could lead to otitis externa.



This figure shows whitish mass in the external auditory canal with evidence of thickening of canal skin. This is caused by candida infection involving the canal.



This figure shows whitish patch over ear drum. This is known as white drum / tympanosclerosis



Retracted drum showing the ossicles

This image classically shows the retraction pocket / adhesive otitis media.

This condition is caused due to middle ear vacuum due to Eustachian tube pathology. The ear drum is found retracted and plastered to the promontory. The thinned out ear drum could be seen to reveal quite a lot of middle ear anatomy. The figure shows all the three ossicles, oval window, round window, pyramid and stapedial tendon.

Features of retracted ear drum:

1. Distortion of cone of light
2. Prominent lateral process of malleus
3. Foreshortening (apparent) of handle of malleus. This occurs because the handle of malleus is pulled medially towards the promontory
4. Mobility of ear drum is diminished / restricted on siegelization
5. These patients have conductive deafness



This is an image of siegles pneumatic speculum

A siegel's pneumatic speculum has an eye piece which has a magnification of 2.5 times. It is a convex lens. The eye piece is connected to an aural speculum. A bulb with a rubber tube is provided to insufflate air via the aural speculum. The advantages of this aural speculum is that it provides a magnified view of the ear drum, the pressure of the external canal can be varied by pressing the bulb thereby the mobility of ear drum can be tested. Since it provides adequate suction effect, it can be used to suck out middle ear secretions in patients with CSOM. Ear drops can be applied into the middle ear by using this speculum. Ear is first filled with ear drops and a snugly fitting siegel's speculum is applied to the external canal. Pressure in the external canal is varied by pressing and releasing the rubber bulb, this displaces the ear drops into the middle ear cavity.



Figure showing keratosis obturans. This is actually a painful condition involving the external auditory canal.

Keratosis obturans¹⁴ is accumulation of desquamated keratin in the external auditory meatus. This should be differentiated from primary auditory canal cholesteatoma which is characterized by invasion of squamous tissue from the external ear canal into a localized area of bone erosion. These patients present with:

1. Severe otalgia
2. Mild to moderate conductive hearing loss
3. Associated sinusitis and bronchitis are rather common
4. Ear canal appears widened

Ear drum perforations:

This should be looked for. For description purposes the ear drum is divided into 4 quadrants.

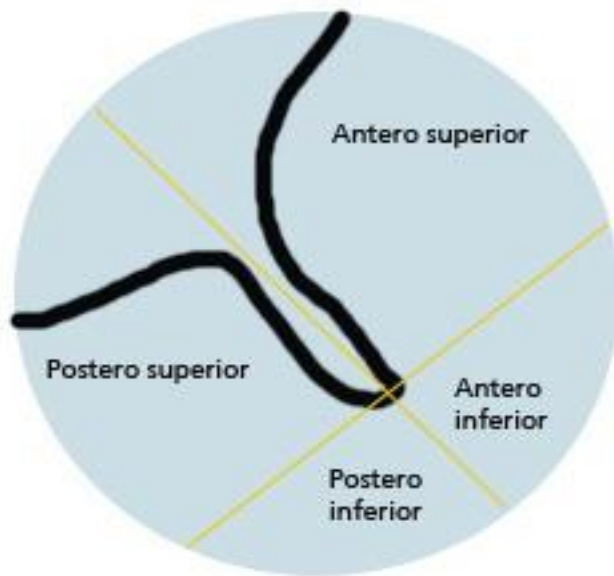


Figure showing the four quadrants of ear drum marked

While describing perforations involving ear drum focus should be paid on:

1. Shape of the perforation. A slit like linear perforation is seen due to trauma. Perforations due to middle ear infections are reniform shaped.
2. Quadrants involved in the perforation
3. Whether it is a large central perforation?
4. Whether it is a subtotal perforation? A subtotal perforation is a large central perforation involving the pars tensa portion of ear drum. The entire pars tensa portion of ear drum is destroyed leaving behind the annulus rim.
5. Whether it is a total perforation of pars tensa? In total perforation of pars tensa the entire pars tensa portion of the ear drum is destroyed including the annulus. It should be stressed that the presence of annulus protects the middle ear cavity from squamous epithelial migration, thereby preventing formation of cholesteatoma. This type of perforations is seen in patients affected with exanthematous fever. Cholesteatoma in these patients is known as secondary acquired cholesteatoma. In secondary acquired cholesteatoma the classic cholesteatomatous sac is not evident, only cholesteatomatous flakes are seen. Cholesteatomatous sac is a characteristic feature of primary acquired cholesteatoma which occurs as a result of attic retraction.

While describing perforations involving pars tensa of ear drum the status of edges of perforation also should be documented. This is important from the therapeutic point of view. If the edges of perforation are fresh and raw as seen in traumatic perforations, wait and watch would help. Spontaneous healing is rather common in these patients. These patients should be discouraged from using ear drops because it could dislodge the clot which attempts to bridge the perforation thereby delay the process of healing.



Figure showing traumatic perforation

In perforations due to chronic infections of the middle ear cavity, the edges of the perforation are rather smooth. The edges could be seen merging with the middle ear mucosa. Spontaneous healing in these patients is rather rare. These patients should undergo myringoplasty.



Figure showing large central perforation involving pars tensa

Perforations involving pars flaccida should be considered sinister. This portion of ear drum doesn't have annulus which protects the middle ear from squamous epithelial migration.



Figure showing perforation involving pars flaccida.

Classification of CSOM has undergone lots of changes. This classification has been proposed taking into consideration the prognostic features of these types of perforations.

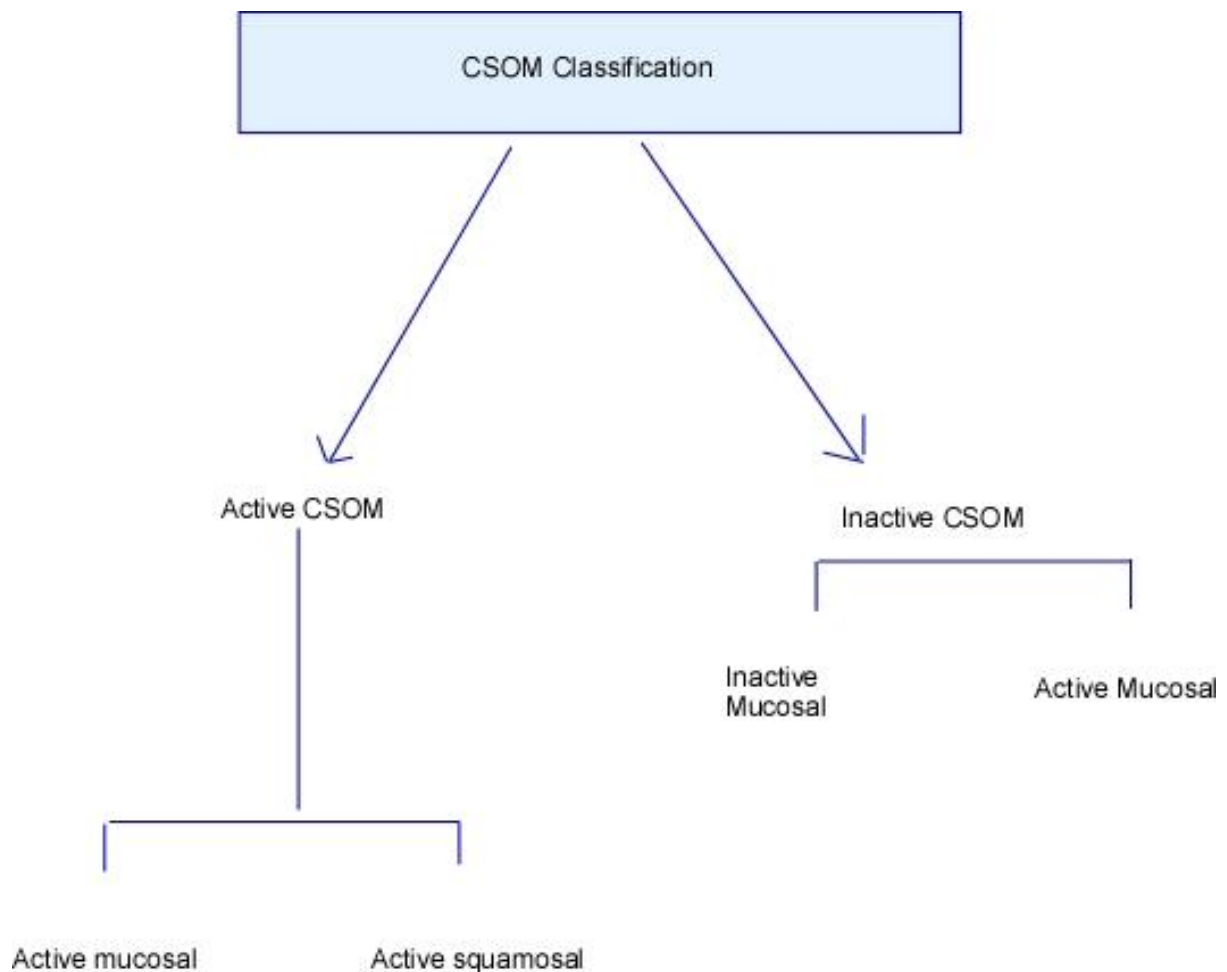


Figure showing the current classification of CSOM

Chronic otitis media simply does not mean accumulation of pus in the middle ear cavity. It can be subdivided into active chronic otitis media (active com) and inactive chronic otitis media (inactive com).

Active COM: In this condition there is inflammation of middle ear mucosa associated with accumulation of pus. There may also be associated mastoiditis. Active COM can be subdivided into active mucosal COM and active squamosal COM.

Active mucosal COM:

Ear drum in these patients will be perforated. The middle ear mucosa may undergo polypoidal changes causing "aural polypi". It is also important to realize that inflammatory changes in this disorder is not confined to the middle ear alone, the whole of the middle ear cleft is involved. Simple closure of the perforation without removal of infected middle ear mucosa and granulations from the mastoid cavity is fraught with failure to control the disease.

Active mucosal COM is often associated with resorption of parts or whole of ossicular chain. This could be due to resorptive osteitis. The ossicles affected typically show hyperemia with proliferation of capillaries and prominent histiocytes. Long process of incus gets eroded commonly, followed by stapes crurae, body of incus and manubrium in that order.

Active squamous COM: This condition is otherwise known as unsafe ear or cholesteatoma. This condition is commonly associated with retraction of pars flaccida / tensa that has retained squamous epithelial debris. There is also associated inflammation of middle ear mucosa, production of pus, and erosion of ossicles. This condition is commonly associated with intracranial complications.

Inactive Chronic otitis media: In this condition the middle ear mucosa is relatively healthy. The mastoid cavity also appears healthy. These patients may slip into active phase rather easily because of the existing pathology. Inactive chronic otitis media can further be subdivided into Inactive mucosal chronic otitis media and Active squamous chronic otitis media.

Inactive mucosal chronic otitis media: This condition is always associated with dry perforation of the ear drum. There is permanent perforation of the pars tensa, but the middle ear and mastoid mucosa are not inflamed. The drum remnant around the perforation is always healthy. The rim of the perforation is thickened due to proliferation of fibrous tissue. Squamous epithelial cells from the external auditory canal do not migrate into the middle ear cavity in this stage because the annulus of the ear drum is intact and it prevents this migration. These patients benefit from myringoplasty.

Inactive squamous epithelial chronic otitis media: These include retraction pockets, atelectasis and epidermisation. Negative middle ear pressure can cause retraction of tympanic membrane. A retraction pocket consists of an invagination into the middle ear space of part of the ear drum. These retraction pockets may be fixed when it is adherent to structures in the middle ear or free when it can move freely medially or laterally depending on the state of inflation of the middle ear. "Epidermisation" is a more advanced type of retraction and it refers to replacement of middle ear mucosa by keratinizing squamous epithelium without retention of keratin debris. The area of epidermisation may involve part or the entire middle ear cavity. Epidermisation often remain quiescent and does not progress to cholesteatoma or active suppuration. So epidermisation per se is not an indication for surgical intervention.

Healed chronic otitis media: In this stage the perforated ear drum has managed to heal itself. Loss of lamina propria or the tympanic membrane due to atrophy or failure of complete healing leads to a 'dimeric' membrane that consists of epidermis and mucosa only. Such thin membrane is more prone to retraction if there is negative middle ear pressure.

Bed side tests for hearing:

Useful bedside test for hearing is performed using a tuning fork. Ideally 3 frequencies are used 256 Hz, 512 Hz, and 1024 Hz. These three frequencies are used because they fall within speech frequency range. An ideal tuning fork should have the following features:

It should be made of a good alloy.

It should vibrate for one full minute.

It should not produce any over tones.

Tuning fork tests are performed to identify whether the patient is suffering from conductive deafness, sensorineural deafness, or mixed deafness. Three tests are performed towards this end. They are 1. Rinne's test, 2. webers test, 3. Absolute bone conduction test / ABC.

Rinnes test: Ideally 512 tuning fork is used. It should be struck against the elbow or knee of the patient to vibrate. While striking care must be taken that the strike is made at the junction of the upper 1/3 and lower 2/3 of the fork. This is the maximum vibratory area of the tuning fork. It should not be struck against metallic object because it can cause overtones. As soon as the fork starts to vibrate it is placed at the mastoid process of the patient. The patient is advised to signal when he stops hearing the sound. As soon as the patient signals that he is unable to hear the fork anymore the vibrating fork is transferred immediately just close to the external auditory canal and is held in such a way that the vibratory prongs vibrate parallel to the acoustic axis. In patients with normal hearing he should be able to hear the fork as soon as it is transferred to the front of the ear. This result is known as Positive Rinne test. (Air conduction is better than bone conduction). In case of conductive deafness the patient will not be able to hear the fork as soon as it is transferred to the front of the ear (Bone conduction is better than air conduction). This is known as negative Rinne. It occurs in conductive deafness. This test is performed in both the ears.

If the patient is suffering from profound unilateral deafness then the sound will still be heard through the opposite ear this condition leads to a false positive Rinne.



Figure showing Rinne's test being performed

Weber's test:

Here again 512 Hz tuning fork is used. The vibrating fork is placed over the forehead of the patient and he is asked to indicate on which side he is hearing the sound. Normally when hearing level is equal in both the ears, it is heard in the middle, in patients with conductive deafness the sound is heard in the left ear. This is known as lateralisation of Weber test. If the patient is suffering from sensorineural hearing loss then the sound is lateralised to the normal ear or the better ear. Hence weber's test must always be interpreted along with the Rinne's test. Weber's test is a sensitive test, it can pin point even a 10 dB hearing difference between the ears.



Figure showing Weber's test being performed

Absolute bone conduction test:

This test is performed to identify sensorineural hearing loss. In this test the hearing level of the patient is compared to that of the examiner. The examiner's hearing is assumed to be normal. In this test the vibrating fork is placed over the mastoid process of the patient after occluding the external auditory canal. As soon as the patient indicates that he is unable to hear the sound anymore, the fork is transferred to the mastoid process of the examiner after occluding the external canal. In cases of normal hearing the examiner must not be able to hear the fork, but in cases of sensorineural hearing loss the examiner will be able to hear the sound, then the test is interpreted as ABC reduced. It is not reduced in cases with normal hearing.

Basic tests for hearing:

For making a basic assessment of patient's hearing the ear opposite to the one tested is masked by occluding it. The patient is asked to repeat random numbers uttered by the examiner. Ideally patient is blindfolded to prevent lip reading. The numbers are uttered at various intensities, quiet whisper, loud whisper, quite voice, loud voice and shout.

Rough estimation of hearing loss would be:

Quite whisper - normal

Loud whisper - 20 - 30 dB

Quite voice - 30 - 45 dB

Loud voice - 45 - 60 dB

Shout - 60 - 80 dB

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